AMENDMENTS TO THE CLAIMS

1-14. (Canceled)

15. (Currently amended) A method of modulating endothelial cell intercellular permeability in a mammal, said method comprising modulating the functional activity of protein kinase Cζ in said mammal wherein up-regulating protein kinase Cζ activity to a functionally effective level up-regulates said endothelial cell intercellular permeability and down-regulating protein kinase Cζ activity to a functionally ineffective level down-regulates said intercellular permeability wherein said modulation of endothelial cell intercellular permeability is achieved by contacting said endothelial cell with a proteinaceous or non-proteinaceous modulation agent which associates with a protein kinase Cζ protein or a protein kinase Cζ nucleic acid molecule.

16. (Currently amended) The method according to claim 15, wherein said intercellular permeability is vascular endothelial cell intercellular permeability or lymphatic endothelial cell intercellular permeability.

17. (Canceled)

18. (Canceled)

19. (Currently amended) The method according to claim 16, wherein said permeability is thrombin-induced vascular endothelial cell intercellular permeability.

20. (Withdrawn) The method according to claim 15, wherein said modulation is up-regulation of protein kinase Cζ activity and said up-regulation is achieved by introducing into said endothelial cell a nucleic acid molecule encoding protein kinase Cζ or functional equivalent, derivative or homologue thereof or the protein kinase Cζ expression product or functional derivative, homologue, analogue, equivalent or mimetic thereof.

21. (Currently amended) The method according to claim 15, wherein said modulation is achieved by contacting said endothelial cell with a proteinaceous or non-proteinaceous modulation agent which modulates transcriptional or translational regulation of the protein kinase Cζ gene.

22. (Withdrawn) The method according to claim 15, wherein said modulation is up-regulation of protein kinase Cζ activity and said up-regulation is achieved by contacting said

endothelial cell with a proteinaceous or non-proteinaceous modulation agent which functions as an agonist of the protein kinase Cζ expression product.

23. (Previously presented) The method according to claim 15, wherein said modulation is down-regulation of protein kinase Cζ activity and said down-regulation is achieved by contacting said endothelial cell with a proteinaceous or non-proteinaceous modulation agent which functions as an antagonist to the protein kinase Cζ expression product.

24. (Withdrawn) The method according to claim 23, wherein said modulation agent is angiopoietin-1 or functional derivative, homologue, analogue, equivalent or mimetic thereof.

25. (Withdrawn) The method according to claim 23, wherein said modulation agent is chelerythrine chloride or bisindoylmaleimide I or functional derivative, homologue, analogue, equivalent or mimetic thereof.

26. (Withdrawn) The method according to claim 23, wherein said modulation agent is a mutant protein kinase Cζ which mutant is characterised by substitution of the threonine residue at position 410 of the activation loop to alanine.

27-48. (Canceled).

49. (Currently amended) The method of claim 9<u>15</u>, wherein said endothelial cell activity is endothelial cell intercellular permeability, and wherein said endothelial cell is contacted with a small molecule antagonist of the PKCζ expression product.

50. (Currently amended) The method of Claim 9<u>15</u>, wherein said modulation agent is selected from the group consisting of a small molecule, an antibody, ~~or~~ <u>and</u> an analogue of the PKCζ expression product.

51. (Currently amended) The method of Claim ~~7~~<u>15</u>, wherein said modulation agent is selected from the group consisting of antibodies, antigens, RNA, ribosomes, DNAzymes, RNA aptamers, and antisense nucleic acids.

52. (New) The method of Claim 15, wherein said endothelial cell intercellular permeability is thrombin-induced, and wherein said method further comprises:

identifying a mammal in need of modulation of thrombin-induced endothelial cell permeability; and

measuring said modulation of thrombin-induced endothelial cell permeability.